Important Facts about Mycotoxins that Every Dairy Producer Should be Aware Of

The goal of the dairy farming industry is to supply dairy processing enterprises with high-quality milk that can be converted into highly nutritious milk products for human consumption. Milk quality is determined by the composition of its nutrients (i.e., fat, protein, lactose, vitamins and minerals), and the level of contaminants or undesirable compounds (*i.e.*, bacterial cells, other cells, and toxins such as aflatoxin M1). Therefore, every dairy farm aims to maximise the level of nutritious components and minimise the levels of contaminants in milk. Many factors on farms are known to influence milk quality. These include dairy cow breed, season, nutrition, management, hygiene, storage, and transport. It is well established that many nutritional factors can contribute to the presence of contaminants and undesired compounds in the milk.

Mycotoxins are toxic metabolites produced by fungi (moulds) which grow on animal feeds, including pasture, forages, cereal grain, by-products and straw. Hundreds of mycotoxins have been identified and one mould species can produce many different types. Feeding mycotoxins contaminated rations (mainly with aflatoxin B1 [AFB1]) can lead to the appearance of mycotoxins (mainly aflatoxin M1 [AFM1]) in milk. Although AFM1 is the main mycotoxin that is transferred from feed to milk, other mycotoxins can be transferred at lower rates and may contribute to reducing the milk quality. Fumonisin B1, α-ochratoxin, T-2 toxin, de-epoxy-DON (DOM), and α-zearalenol can be transferred into milk when highly contaminated rations are fed. In addition, feeding mycotoxin contaminated rations may reduce fat and protein content, and increase somatic cell counts and bacterial load in milk; all of which contribute to significantly reducing the milk quality.

Negative Effects of Mycotoxins in Ruminants

Chronic exposure: Quite often problems are due to low levels of mycotoxins and may be expressed as just minor increases in "common cow problems", especially with freshly calved cows.

Acute exposure: Consumption of high levels of mycotoxins may give rise to symptoms including abrupt drops in milk production and feed intake, abortions, lameness and, in the most severe cases, mortality.

Diagnosis of Mycotoxin Issues

Symptoms are often either sub-clinical or are non-specific and easily confused with other disorders. Poor response to veterinary therapy is frequently an indicator that mycotoxins are implicated in a condition. Feed can be tested for mycotoxins but the accuracy of sampling and the cost of routine testing limit its practicality at farm-level.

Mycotoxin Threat to Ruminants?

It is extremely difficult to identify when mycotoxins are causing poor health and performance. Some mycotoxins, such as zearalenone, predominantly affect reproduction and are relatively easy to identify. Mycotoxins that can cause acute intoxications and dramatic changes in milk production and animal health status at high levels can be identified much more easily. Unfortunately, the most common and most difficult problem to identify occur when rations contain low levels of the mycotoxins and the health effects are sub-clinical. The presence of mycotoxins in feed is often connected with an increase in the incidence of metabolic disorders such as ketosis, retained placentas, displaced abomasums, mastitis, metritis, lameness, elevated somatic cell counts (SCC) and, consequently, slightly decreased milk production. Sub-clinical mycotoxicoses reduces profitability by lowering milk production and increasing expenses from additional veterinary therapies.

Mycotoxins can be the primary agents causing acute health or production problems in a dairy herd, but are more likely to be a contributing factor to chronic problems including a higher incidence of diseases, poor reproductive performance, or suboptimal milk production. Mycotoxins exert their effects through four primary mechanisms: intake reduction or feed refusal, reduced nutrient absorption and impaired metabolism, alterations in the endocrine and exocrine systems, and suppression of the immune system. Recognition of the impact of mycotoxins on animal production has been limited by the difficulty in diagnosis. Symptoms are often non-specific and can be the result of a progression of effects, making a diagnosis difficult or impossible because of the complex clinical results with a wide range of symptoms.

What Do We Need to Know about Aflatoxins in Ruminants

Aflatoxicosis is the disease caused by the consumption of high levels of aflatoxins. At low levels of intake, usually there are no visual symptoms of aflatoxicosis, and as such the problem is often unnoticed. However, high concentrations of aflatoxins, or prolonged exposure at low levels, cause visual symptoms in cattle, and especially in young calves. Beef and dairy cattle are more susceptible to aflatoxicosis than sheep and horses, whereas young animals of all species are more sensitive to the effects of aflatoxins than mature animals. On the other hand, pregnant and growing animals suffer from aflatoxicosis less than young animals, but more than mature animals kept at maintenance (for example, breeding males).

Feed refusal, reduced growth rate, and decreased feed efficiency are the predominant signs of chronic aflatoxin poisoning. In addition, weight loss, rough hair coat, and mild diarrhoea may be observed in affected animals. Anaemia, along with bruises and subcutaneous haemorrhages are also frequent symptoms of aflatoxicosis. This disease may also impair reproductive efficiency, including abnormal oestrous cycles (too short or too long) and increased abortions. Other symptoms include impaired immune system response, increased susceptibility to other diseases, and rectal prolapse.

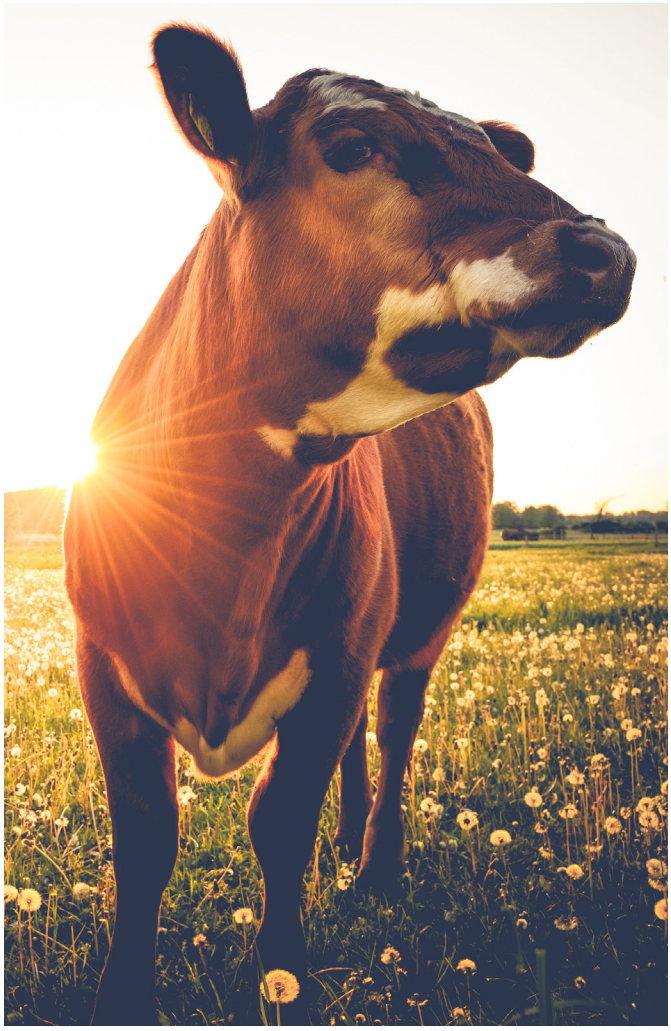
The diagnosis of aflatoxicosis is often difficult because of the variation in clinical signs, gross pathological conditions, and the presence of secondary infectious diseases due to the suppression of the immune system. In addition, under commercial conditions, more than one

mycotoxin may be present in any contaminated feed, and this makes definitive diagnosis of aflatoxicosis quite difficult. The effects of aflatoxin contamination as the disease progresses depend upon the severity of caused liver damage. Thus, once overt symptoms are noticed, the prognosis is usually poor. Treatment should be directed at the severely affected animals in the herd, and measures should be taken to prevent further poisoning. Unfortunately, most lactating cows positive for aflatoxins in milk will not exhibit strong visual symptoms, and as such, prevention is always the best way to tackle this problem.

With most mycotoxins being carcinogenic to animals and humans, there is a wide legislation framework regarding their monitoring in the food supply chain. Aflatoxin B1 is the most carcinogenic natural compound known. Aflatoxin M1 is the natural metabolite of aflatoxin B1 and it has a high carry-over rate to animal products such as milk. Fresh milk is regularly checked for aflatoxin M1; concentrations of M1 above 0.05 µg/kg in the EU, or 0.5 µg/kg in the US, are considered undesirable and such milk cannot be used for products that go into the human food chain. Contaminated milk must be discarded, and apart from the cost of lost milk revenue, the dairy producer must also suffer the cost of properly disposing of the contaminated milk! The carry-over rate of aflatoxins from contaminated feed into milk in dairy cows is considered to average 1–2%. However, in high-yielding cows, which consume significant amounts of concentrated feeds, the carry-over rate of aflatoxin M1 into milk can reach 6.2%.

Legal limits / Advisory guidelines*		
Mycotoxin	**EU**	**FDA**
Aflatoxin B1	5 ppb	20 ppb
Deoxynivalenol	5 ppm*	5 ppm*
Fumonisins	50 ppm*	15 ppm*
Ochratoxin A	250 ppb*	ND
Zearalenone	500 ppb*	ND
EU = European Union, FDA – US Food and Drug Administration ND = not determined		

Table 1: EU and FDA legal limits for aflatoxins and advisory guidelines on safe levels for other mycotoxins in finished feed for dairy cattle
**Commission directive, 2003; Commission recommendation, 2006*



Can Rumen Effectively Detoxify Mycotoxins?
The rumen has long been considered relatively resistant to mycotoxins because rumen microflora was assumed to naturally detoxify mycotoxins. However, stressed dairy cows such as those that are sick and/or lactating may have an increased rumen passage rate or overwhelmed rumen microflora and therefore not able to denature all of the toxins in contaminated feed. The same is for calves which are more susceptible to mycotoxins, as their rumens are not completely developed. A major factor in the absorption of mycotoxins in ruminants is rumen fermentation. Table 2 provides a summary of the degree of rumen mycotoxin bio-conversion and transfer to milk.

Mycotoxin	Main product of rumen metabolism	Reduction of biological potency	Estimated carry-over rates to milk
Aflatoxin B1	Aflatoxicol	Minor	0–12,4 µg/L
	Aflatoxin M1	Minor	2,0–6,2%
Fumonisin B1	Unchanged	Unchanged	0–0,05%
Ochratoxin A	α-Ochratoxin	Significant	ND
T-2 toxin	Various	Significant	0,05–2%
DON	De-epoxy DON (DOM-1)	Significant	DON: 0,0001–0,0002%
			DOM: 0,0004–0,0024%
Zearalenone	α-Zearalenol	None	0,06–0,08%
ND = Not determined; DOM-1 = deoxynivalenol metabolite-1			

Table 2 - Rumen bioconversion and transfer of mycotoxins from feed to milk (Adapted from Fink-Gremmels et al., 2008)

Ruminal degradation of mycotoxins helps to protect the cow against acute toxicity, but may contribute to chronic problems, associated with long-term consumption of low levels of mycotoxins. Ruminal degradation of mycotoxins may have helped mask mycotoxin effects in dairy cows which were recognised in recent years, as production stresses have increased and as the industry has paid more attention to management details.

Mycotoxins Exert their Effects through Several Means:

1. Reduced intake or feed refusal
2. Reduced nutrient absorption and impaired metabolism
3. Altered endocrine and exocrine systems
4. Suppressed immune function
5. Altered microbial growth

Recognition of the impact of mycotoxins on animal production has been limited by the difficulty of diagnosis. The progression and diversity of symptoms are confusing, making diagnosis difficult (Hesseltine, 1986; Schiefer, 1990). The difficulty of diagnosis is increased due to limited research, occurrence of multiple mycotoxins, non-uniform distribution, interactions with other factors, and problems of sampling and analysis. Because of the difficulty of diagnosis, the determination of a mycotoxin problem becomes a process of elimination and association.
Certain basics can be helpful (Schiefer, 1990):

1. Mycotoxins should be considered as a possible primary factor resulting in production losses and increased incidence of disease.
2. Documented symptoms in ruminants or other species can be used as a general guide to symptoms observed in the field.
3. Systemic effects, as well as specific damage to target tissues, can be used as a guide to possible causes.
4. Post-mortem examinations may indicate no more than gut irritation, edema, or generalised tissue inflammation.
5. Because of the immune-suppressing effects of mycotoxins, increased incidence of disease or atypical diseases may be observed.
6. Responses to added dietary adsorbents or dilution of the contaminated feed may help in diagnosis.
7. Feed analyses should be performed, but accurate sampling is a major problem.

Symptoms vary depending on the mycotoxins involved, and their interactions with other stress factors and animals may exhibit few or many of a variety of symptoms. The more stressed cows, such as fresh cows, are most affected; perhaps because their immune systems are already suppressed.

Economically Most Important Mycotoxins and their Effects in Ruminants

Deoxynivalenol (DON)

- Dairy cattle consuming diets contaminated primarily with DON (2.5 ppm) have responded favourably (1.5 kg milk, $P<.05$) to the dietary inclusion of a mycotoxin binder, providing circumstantial evidence that DON may reduce milk production (Diaz *et al*., 2001).
- Field reports help substantiate this association (Gotlieb, 1997). Results from a Canadian study using six first-lactation cows per treatment during mid lactation (average 19.5 kg milk) showed that cows consuming DON-contaminated diets (2.6 to 6.5 ppm) tended ($P<0.16$) to produce less milk (13% or 1.4 kg) than did cows consuming clean feed (Charmley *et al*., 1993).
- DON has been associated with altered rumen fermentation (Seeling *et al*., 2006) and reduced flow of utilisable protein to the duodenum (Danicke *et al*., 2005). Fusaric acid interacts with DON to cause the vomiting effects, which earlier were attributed to DON alone and resulted in use of the trivial name of vomitoxin for DON (Smith and MacDonald, 1991).

Remark: *It is believed that DON serves as a marker, indicating that feed was exposed to a situation conducive for mould growth and possible formation of several mycotoxins.*

T2–toxin

- In dairy cattle, T2-toxin has been associated with gastroenteritis, intestinal haemorrhages (Petrie *et al*., 1977) and death (Hsu *et al*., 1972).
- Dietary T2-toxin at 640 ppb for 20 days resulted in bloody faeces, enteritis, abomasal and ruminal ulcers, and death (Pier *et al*., 1980).
- Weaver *et al*. (1980) showed that T2-toxin was associated with feed refusal and gastrointestinal lesions in a cow.
- Kegl and Vanyi (1991) observed bloody diarrhoea, low feed consumption, decreased milk production, and absence of oestrous cycles in cows exposed to T2-toxin.
- Serum immunoglobulins and complement proteins were lowered in calves receiving T2-toxin (Mann *et al*., 1983). Gentry *et al*. (1984) demonstrated a reduction in white blood cell and neutrophil counts in calves.
- McLaughlin *et al*. (1977) demonstrated that the primary basis of T2-toxin reduced immunity is reduced protein synthesis.

Zearalenone (ZEA)

- In a study with heifers receiving 25 ppm of ZEA, conception rate was depressed about 25% (Weaver *et al*., 1986).
- Several case reports have related ZEA to oestrogenic responses in ruminants including abortions (Khamis *et al*., 1986). Symptoms have included vaginitis, vaginal secretions, poor reproductive performance, and mammary gland enlargement of virgin heifers.
- In a study (Coppock et al., 1990), diets with about 660 ppb ZEA and 440 ppb DON resulted in poor consumption, depressed milk production, diarrhoea, increased reproductive tract infections, and total reproductive failure.
- Towers *et al*. (1995) have measured blood ZEA and metabolites ("zearalenone") to estimate ZEA intake. Dairy herds with low fertility had higher levels of blood "zearalenone". Individual cows within herds examined by palpation and determined to be cycling had lower blood "zearalenone" levels than did cows that were not cycling. In this study, reproductive problems in dairy cattle were associated with dietary ZEA concentrations of about 400 ppb.

Fumonisins

- Osweiler *et al*. (1993) fed 18 young steers 148 ppm of fumonisin in 31 days. There were mild liver lesions found in two of six calves, and the group had lymphocyte blastogenesis and elevated enzymes indicative of liver damage.
- Dairy cattle (Holsteins and Jerseys) fed diets containing 100 ppm fumonisin for approximately seven days prior to freshening and for 70 days thereafter demonstrated lower milk production (6kg cow/day), explained primarily by reduced feed consumption (Diaz *et al*., 2000).
- Fumonisin carry-over from feed to milk is thought to be negligible (Scott *et al*., 1994).

α-Ochratoxin (OTA)

- In cattle, OTA is rapidly degraded in the rumen (to α-Ochratoxin) and thus thought to be of little consequence unless consumed by young pre-ruminant calves (Sreemannarayana *et al*., 1988).
- With high-grain diets, less of the dietary ochratoxin may be degraded in the rumen and thus be more toxic in those situations (Hohler *et al*., 1999).
- Mouldy alfalfa hay containing *Aspergillus ochraceus* was implicated as producing OTA associated with abortions in cattle (Still *et al*., 1971).
- OTA in mouldy forage has also been implicated in cattle deaths (Vough and Glick, 1993).

Ergot Alkaloids

- Ergotism primarily causes a gangrenous or nervous condition in animals. Symptoms are directly related to dietary concentrations and include reduced weight gains, lameness, lower milk production, agalactia and immune suppression (Robbins *et al*., 1986).
- Fescue infected with *Neotyphodium* sp. or *Epichloe* sp. may contain toxic alkaloids associated with "fescue toxicity" (CAST, 2003). Symptoms include lower weight gains, rough hair coat, increased body temperature, agalactia, reduced conception, and gangrenous necrosis of the extremities such as the feet, tail and ears.

Aflatoxins

- Symptoms of acute aflatoxicosis in mammals include: inappetance, lethargy, ataxia, rough hair coat, and pale, enlarged fatty livers. Symptoms of chronic aflatoxin exposure include reduced feed efficiency and milk production, jaundice, and decreased appetite. Aflatoxin lowers resistance to diseases and interferes with vaccine-induced immunity (Diekman and Green, 1992).
- In beef cattle, Garrett *et al*. (1968) showed an effect on weight gain and intake with diets containing 700 ppb aflatoxin, but if increases in liver weights are used as the criteria for toxicity, 100 ppb would be considered toxic to beef cattle.
- Production and health of dairy herds may be affected at dietary aflatoxin levels above 100 ppb, which is considerably higher than the amount that produces illegal milk residues (Patterson and Anderson 1982).
- Guthrie (1979) showed when lactating dairy cattle in a field situation were consuming 120 ppb aflatoxin, reproductive efficiency declined, and when cows were changed to an aflatoxin free diet, milk production increased over 25%.
- Applebaum *et al*. (1982) showed milk production was reduced in cows consuming impure aflatoxin produced by culture, but production was not significantly affected by equal amounts of pure aflatoxin.

Economic Evaluation of Mycotoxin Deactivators in Dairy Cows

Production losses due to mycotoxin contamination are clearly subject to a great number of factors and uncertainties. The losses are hugely variable in time and difficult to estimate. However, the effects of the contamination are often significant and can be long-lasting. The economic impact of mycotoxins is difficult to estimate even after an outbreak of mycotoxicosis. The most important losses are probably those associated with long-term under-performance. Estimates of this can be made on the basis of the information provided above. Thus, a simple simulation model was developed that allows for the estimation of production and financial losses due to the long-term sub-clinical impact of mycotoxins in dairy cattle.

The following assumptions were made:

- No change in dry matter intake or loss in milk production volume.
- A decrease of 0.4% - point in milk fat and 0.1% - point in milk protein.
- No penalising change in SCC, thus assuming almost ideal sanitary conditions of cows.
- An increase in calving interval of 60 days and an increase in inseminations by 10% along with an increase in veterinary cost of 10%.
- Application of an efficient mycotoxin deactivator restores losses by 80%.

Under these assumptions, the model predicts that on a herd basis, mycotoxin contamination will cause losses in milk income of approximately 12% and that the addition of an efficient mycotoxin deactivator will restore losses to just 3% under the income level achieved in the absence of mycotoxins. Total farm revenue changed with similar percentages but variable costs or the operation costs increased by 3% in the presence of mycotoxins. The annual return over variable costs decreased from 14.5 to 7.6% due to the presence of mycotoxins. The cost of the mycotoxin deactivator for a continuous treatment throughout lactation and dry period was estimated at $ 28/cow. The application of this mycotoxin treatment led to an improvement in returns over variable cost to 12.3% due to an improvement in revenue of $ 225/cow. Consequently the return on investment (ROI) of the use of a mycotoxin treatment is approximately 7:1. The assumptions associated with these simulations are considered to be rather close to the current US operational conditions. The model can be adapted to other economic situations — for instance those applicable to the EU, Middle East or Latin America. However, following a number of simulations, it appears that the economic returns of mycotoxin deactivators under conditions where contamination is suspected will easily be equal or superior to the rather conservative estimates obtained with these analyses (Van Eys *et al*., 2016).

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